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ON BREATHLESSNESS, ESPECIALLY IN RELATION
TO CARDIAC DISEASE.

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GENTLEMEN,

LET me begin by thanking you and your President for the honour you have done me in asking me to address you. The subject I have chosen for my address is that of breathlessness, and especially breathlessness dependent upon weakness or disease of the heart. I have been induced to select this subject by my recollection of a case which I saw several years ago with Dr. Waterhouse in this district. Moreover, I thought I knew enough about the subject to write a paper about it with ease, and it is only after working at it for several months, reading up the literature in monographs, in four different languages, considering with especial care the work of my friend Professor Angelo Mosso, and discussing respiration in general and breathlessness in particular with my friend Professor Hugo Kronecker, that I find that the subject is one of the most difficult in the whole of physiology or pathology. I must warn you, therefore, that the conclusions at which I have arrived, and which I am now going to put before you, are not to be regarded as an expression of generally accepted facts, but only as the opinions at which I have arrived myself after careful study. Any value they may have depends on their being founded, not on laboratory work alone, nor on clinical work alone, but on both combined. In the case which, as I have just mentioned, I saw, not very far from the place where I am now speaking, that breathlessness was the most prominent symptom, and I diagnosed the patient to be suffering from atheroma of the right coronary artery. To some this diagnosis might seem to be fanciful, but, as I will

¹ Address delivered at a Meeting of the Willesden and District Medical Society.

presently show, it was almost the only one possible to make, and post-mortem examination proved it to be correct. The patient, Mr. B., was a gentleman of 67 years of age, who had been a good deal in the Colonies. I had seen him several times on his visits home. When he returned finally to this country and came to consult me, I was at once struck by his deep and hurried breathing as he walked towards me from the door of my consulting room. I thought I might find something wrong with his lungs, but on examination I found that they were on the whole healthy, the only abnormal conditions to be noted being that the respiratory sounds were exaggerated over both fronts and backs and there was slight crepitation over the lower part of both backs. There was a diastolic murmur indicating some aortic regurgitation, and a very faint systolic murmur at the apex not propagated to the axilla. There was a slight trace of albumen in the urine, of which the specific gravity was 10.28. The appetite was poor and there was some dyspepsia. On considering the causes to which his shortness of breath might be due, it was easy to eliminate the lungs because they were on the whole healthy, the air passed freely in and out of them, and they were doing more work than usual. Notwithstanding the slight trace of albumen, the condition of the kidneys might be regarded as good, for the specific gravity of the urine was unusually high, so that it was clear they could eliminate a large proportion of solids. Having excluded the lungs and the kidneys as causes of his breathlessness, there remained only the heart. The most likely condition to produce the symptom was general fatty degeneration of this organ, and I expected to find weak sounds and a feeble impulse. To my astonishment, however, the sounds were loud, and the apex beat was not only lower down and further out than usual, but it was strong and heaving, showing that the cardiac enlargement was due to hypertrophy and not merely to dilatation. The pulse was strong and regular, and yet his breathlessness was evidently of cardiac origin. I therefore concluded that although the left ventricle was strong and somewhat hypertrophied, yet the right ventricle was so weak as to be unable to properly perform its work of driving the blood through the lungs. The most likely cause of such weakness is fatty degeneration, consequent upon imperfect supply of blood to the ventricle, and I was therefore

forced to diagnose atheroma of the right coronary artery. At the post-mortem examination we found, as I had diagnosed, atheroma of the right coronary artery, but we did not find it exactly of the kind that I had expected. The left ventricle was large, powerful, and well nourished. It was nearly three-quarters of an inch thick, and the muscular structure seemed quite healthy. The right ventricle, on the contrary, was barely a quarter of an inch thick, and so soft and friable that with a slight push the finger went more readily through it than through a sheet of blotting-paper. I expected to find the atheroma round the mouth of the right coronary artery at the point where it left the aorta, and I was therefore a good deal astonished, when the aorta was opened, to find its interior quite smooth and free from atheroma. On slitting up the coronary artery, however, as it ran along the interventricular groove, we found all the branches passing from it to the right ventricle almost occluded by atheroma.

It seems to me that this case is interesting physiologically, as well as pathologically, for it shows very clearly the part played by the heart in the function of respiration. In order to have the blood aërated we require (1st) that fresh air shall freely enter the lungs so as to aërate the blood, and (2nd) that the blood shall flow readily through the lungs, so as to be exposed in sufficient quantity to the air, before it enters the general circulation and goes to aërate the tissues. By stopping either process asphyxia may be produced. By interfering with either process breathlessness may be produced.

I have here used the word breathlessness purposely because its meaning is somewhat vague, and it may be used in two different senses, between which I wish now to differentiate. It is sometimes used to signify increased breathing, and at other times distressed or painful breathing. These conditions are so nearly allied that the first, or increased breathing, readily passes into the second, or painful breathing, and yet the two are essentially distinct. The first, increased breathing, is sometimes called *polypnœa*, because the respirations, as a rule, are quicker as well as deeper; but I think a better term is *hyperpnœa*, because this term signifies increased breathing without taking into consideration whether the respirations are more numerous or simply deeper. A short time ago, on the beach at Margate, I saw a little girl playing at ball. As she

ran hither and thither over the sand, her long hair streaming out behind her, her cheeks glowing, and her eyes gleaming, her respirations were both rapid and deep. But there was no trace of distress in the child's breathing. She had hyperpnœa, but it was *eupnœa*, or pleasant breathing, not *dyspnœa*, or painful breathing, for the expression of her face, her gestures, and the cries she uttered from time to time were all indicative of perfect delight and of the full enjoyment of health and well-being. One could see that the respirations were both rapid and deep, and if I had been able to feel her heart I have no doubt I should have found it beating quickly and strongly ; but although both heart and lungs were acting more than usual they were not acting beyond the bounds of health, and their movements were in perfect co-ordination with each other and with the muscular efforts which the child was making. She was enjoying herself to the utmost, and there was no trace whatever of the distress to which we give the name of dyspnœa. Although her respiratory apparatus was acting excessively, the child was quite unconscious of the fact, and the unusually great action of the heart and lungs was only associated in her mind with an unusual sense of pleasure and well-being. It is when the heart and lungs fail to aërate the blood sufficiently, in spite of all their efforts, that the person becomes conscious of painful breathing or dyspnœa.

Such imperfect aëration as leads to dyspnœa may occur either (*a*) from the air being hindered in its free passage to and from the lungs, or (*b*) from the blood being hindered in its free circulation through the lungs. Both these factors may act conjointly and probably very often do. Indeed interference with the passage of air into or from the lungs appears to have stagnation of blood in the pulmonary vessels as a consequence, for in those cases of asphyxia where death is not due to shock, the lungs are engorged and the right side of the heart distended. A similar condition appears to occur in life during dyspnœa, as I think an observation I once made upon myself appears to show. When crossing the Theodule Pass, in Switzerland, in 1878, my guides began to walk rather quickly. I had thrown off my coat, and my shirt was open at the breast, so that I was able to feel the heart without any trouble. With the increased pace I began to feel a sense of distress and oppression at the

chest. On putting my hand over the spot where the apex beat ought to have been, I was astonished to find that it had disappeared and that marked epigastric pulsation had taken its place. I called to the guides to walk more slowly, and putting my hand upon my breast I found that as the distress diminished the epigastric pulsation disappeared, and I could again feel the apex beat in its normal situation. It is evident from this observation that during the extra exertion the right side of my heart had been unable to send on the blood through the lungs as quickly as it was received from the veins, and that consequently the right ventricle had become dilated (*cf.* Fig. 4, p. 13). This experience has led me to think that the sensation of dyspnœa is probably of cardiac rather than pulmonary origin, and I think it is only when the right heart begins to fail to empty itself that the feeling of dyspnœa comes on. The forced respirations by which one tries to relieve it are I think beneficial, not merely by bringing more air into the lungs, but by a kind of mechanical massage to the heart, as was shown years ago by my friend Professor Hugo Kronecker,¹ and illustrated in the accompanying diagrams (Figs. 1 and 2). It is quite extraordinary how many



Fig. 1. *Diagram of a transverse section of the thorax during inspiration and cardiac systole. It shows the tendency to the formation of a vacuum in the pleural and pericardial cavities into which lymph flows. Air is also drawn into the lungs, and blood is sucked into the auricles from the abdominal veins.*

instances are on record of men and animals having fallen dead, not during the period of violent exertion, but just when it was

¹ Kronecker and Heinrichius: *Abhandlungen der Königl. Sachsischen Gesell. d. Wiss.*, 1888, Bd. XIV., p. 427.

over. A short time ago I was at Fribourg, in Switzerland, and saw a huge linden tree, which is said to have grown from the twig which the messenger held in his hand when he brought to his fellow citizens the news of their victory over Charles the

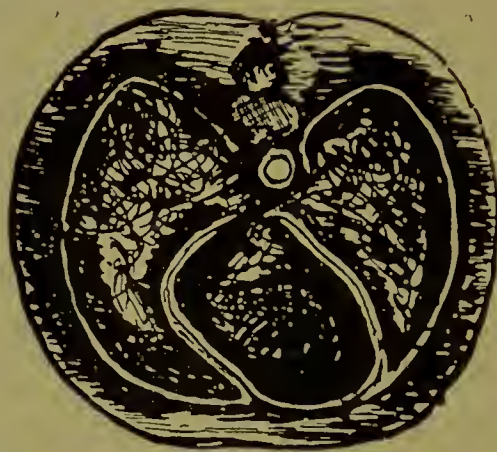


Fig. 2. *Diagram of a transverse section of the chest during expiration and cardiac diastole, showing the pressure of the walls of the pleural and pericardial cavities against each other, so that lymph is pressed out into the lymphatics.*

Bold at Morat. He had run all the way from the battlefield to the town, and was only able to cry "Victory!" when he fell dead. There are many other examples of the same sort, but the best known is that of the Greek who brought to Athens the news of the victory at Marathon, and who fell dead as soon as he had delivered his message. There are numerous accounts also of horses falling dead at the foot of the scaffold after having been ridden hard to bring a reprieve to a condemned criminal. At one time I used to think how fortunate it was that the horses had lasted so long as to reach the scaffold, but now I think that if the reprieve had simply been handed to an official and the horses had not been pulled up quickly, but simply allowed to slacken their speed, they would not have died at all.

Once I nearly lost a train and was obliged to rush up a long flight of steps at the station as quickly as I could with a portmanteau in my hand. I just got into the carriage as the train began to move, and the sensation of dyspnoea that I then had was most distressing, so that I put my head out of the window and took long deep breaths in order to relieve it. It was

during this experience that I began to feel that if, instead of having been obliged to remain quietly in the train after my violent exertion, I had been able to run a little further and slow my pace gradually I should not have had anything like the same distress.

In dyspnœa we must remember that there are two conditions, the subjective and the objective one. In my own case, when I entered the railway carriage there was the subjective sensation of distress and suffocation which I felt, and there was the panting-breathing which my fellow-passengers could observe as well as myself. In considering the subject of dyspnœa we must carefully distinguish between these two conditions, the subjective sensations and the objective phenomena, and we must carefully bear in mind that the relationship of these two factors may vary very considerably.

The causation of breathlessness is very complicated, and at the same time it is most important that it should be thoroughly understood. I think it may, therefore, be worth while to illustrate it by a reference to fatigue in which, as well as in breathlessness and pain, we have a subjective and objective part.

The objective part has been carefully studied in an isolated muscle taken from a frog's leg. When this is kept contracting for a length of time by electrical stimulation the contractions become gradually smaller and feebler, and at length cease entirely, however great the stimulus may be. At first sight it might be supposed that this exhaustion of the muscle is merely due to the force-giving substances it contains being used up, just as a locomotive will cease running when its supply of coal becomes exhausted. But this is only partially the case. The exhaustion is chiefly due to the accumulation of waste products in the muscle which paralyse it, just as an accumulation of ash in the furnace of a locomotive would choke it up and prevent its action. When these waste products are removed by massage of the muscles, or still more efficiently by washing them out by means of artificial circulation, the paralysis disappears, and contractility returns. Such is, broadly sketched, the process of muscular exhaustion in the muscles.

We have now to consider the sensation of fatigue which

is a function of the central nervous system. Like pain, it is a danger-signal. Its object is to give due warning of the condition of the muscles so as to save them from being exhausted, and it comes on, as a rule, long before exhaustion occurs. Like pain, it usually has a peripheral origin; but, like pain, it may be entirely central, and just as excessive pain may be felt by an hysterical woman in a perfectly healthy knee-joint, so excessive fatigue may be felt in the limbs, although the muscles themselves are perfectly healthy and capable of a great amount of exertion. "Constitutionally tired," or, as their friends term them, "lazy" people, are even more common than hysterical people; but a sudden powerful mental stimulus will generally waken lazy people up, and enable them to undergo such exertions as to astonish those who have only known them under ordinary conditions. The same thing occurs even when the muscles are really in a condition of considerable exhaustion. Troops on the march, apparently exhausted and hardly able to drag one foot after the other, will sometimes pull themselves together, and step out as briskly as if they were completely fresh, when they learn that the enemy is near, and there is a prospect of an immediate engagement.

The sensation of fatigue may also be removed by certain drugs which act upon the nervous system. The best known of these is, perhaps, cocaine. The coca leaves have been long used by the natives of Peru to prevent fatigue when making long journeys on foot. Tea, coffee, beef-tea and tobacco have a similar power of lessening fatigue, though perhaps not to the same extent, and so has alcohol, though its effects are only temporary, and are usually succeeded by greater exhaustion. It is possible that these drugs may have a slight effect upon the muscles, but such effect is small compared with the action they exert upon the central nervous system.

As I wish to have the ground thoroughly cleared for the consideration of dyspnœa, I trust you will excuse me if I again recapitulate what I have said in regard to fatigue. There are two distinct conditions in fatigue: one is exhaustion of the muscles themselves, and the other is a sensation of fatigue in the central nervous system. The sensation of fatigue can be removed by acting either peripherally on the muscles or

centrally on the brain. By proper massage of the muscles the waste products in them can be removed, their contractility restored, and, the peripheral cause of fatigue being gone, the sensation of fatigue usually ceases. But the sensation of fatigue, which is felt in the brain, can be removed by lessening the excitability of the sensory nerve-centre to peripheral stimuli, either by the use of drugs or by mental excitement, even though the muscles remain fatigued. Now dyspnœa, like pain and like fatigue, consists of two parts, the peripheral condition and the central sensations, and although they generally bear a definite relationship to one another, they do not always do so. Just as a lazy man may feel tired, although examination of his muscles cannot show the least defect in them, and as an hysterical woman may feel pain in her joints although they are perfectly sound, so a person may feel considerable dyspnœa, although no objective cause can be discovered. Thus one sometimes sees a lady, when sitting quietly in a room, suddenly complain of shortness of breath and a feeling of stifling, and insist upon the window being opened, sometimes to the great annoyance of her neighbours, when to the senses of everybody else the room appears to be perfectly ventilated. In such a case there may be some alteration in the action of the heart, and in the pulmonary circulation, which initiate the feeling of suffocation, but yet this feeling is chiefly of central origin, and is out of proportion to the peripheral condition. Sometimes, on the other hand, you may see patients who have marked hyperpnœa from organic disease, and who yet appear to be unconscious of the fact, but in such cases a very little exertion is usually sufficient to bring on distress and give rise to dyspnœa. In some instances we find the patients make no complaint of dyspnœa, even though lividity of the lips and lobes of the ears plainly shows that the blood is insufficiently aërated. In such instances, as well as in that of the lady craving for fresh air, we have a want of relationship between the peripheral condition and the central sensation, but they are of different kinds in the two cases, there being in the cardiac patient an *anæsthesia*, and in the nervous lady a *hyperæsthesia* of the cerebral centre for respiratory sensations, wherever that centre may be. Perhaps some of you may

think I have dwelt upon this point at too great length, but I am anxious to emphasise it, because it has an important bearing on treatment, inasmuch as we have sometimes to direct our attention chiefly to the peripheral, and at others to the central condition.

Here again, I must draw attention to a possible mistake in the meaning of the word "central," because it may include both the medulla oblongata and the cerebrum as distinguished from the lungs and heart. It is in the cerebrum alone that sensation is perceived, while the medulla, lungs, and heart will maintain the circulation and respiration after the cerebrum has been completely removed. The popular expression of "bellows" in regard to the breathing apparatus is very descriptive, because air is alternately sucked into the chest and expelled from it by the thoracic walls and diaphragm in much the same way as in a pair of bellows. The muscular movements which effect this receive their innervation from the respiratory centre in the medulla oblongata, or more properly from the respiratory centres, because there is both an inspiratory and an expiratory one. The inspiratory one is in constant action, the expiratory one being usually passive,¹ but the expiratory centre is also called into play when any extra demand is made upon the respiration. The action of the respiratory centre is of a so-called automatic character, *i.e.*, the action depends upon changes in the nerve cells which form the centre, changes which are independent of circulation and reflex action, but are largely influenced by the condition of the blood flowing through the centre, so that, according to Fredericq, when the blood is very venous² the centre is excited, and, when the blood is well aerated, the irritability of the centre is diminished. The rhythm of the respiratory movements is, however, regulated almost entirely by reflex impressions passing to the centre through the vagus nerve from the lungs and heart, and through the trigeminus from the upper respiratory passages, though the posterior corpora quadrigemina have

¹ Marckwald: *The Movements of Respiration*. 1888, London: Blackie, pp. 60 and 117.

² I have preferred the word venous in order to avoid discussion of the question how far the stimulating power of such blood over the respiratory centre depends on absence of oxygen, or presence of carbonic acid, or presence of other substances.

also a regulating power and can replace the action of the vagi when these are divided.¹ It is probable that both the respiratory stimuli, which act centrally on the nervous system, and those which act peripherally on the lungs and heart, are chemical rather than mechanical, and that they consist of substances whose stimulating action is diminished or destroyed whenever they are fully oxidised. When these substances are produced too quickly or in circumstances which lessen their oxidation, we get hyperpnœa or dyspnœa according to the quantity of the stimulant present. A little extraordinary exertion appears to produce them more quickly than they can be destroyed, so that a person who has run upstairs may palpitate and pant for several minutes after the exertion has ceased. When the oxidising power of the blood is diminished, as in anæmia, less exertion is sufficient to produce the panting, and it is apt to last longer after it has been produced. We do not at present know exactly what these stimulating substances are, nor where they are all produced, but many of them are probably produced in the muscles, for Kronecker found that, if one muscle be tetanized, the whole body may be paralysed by its waste products, and Mosso found that the blood of fatigued animals, when injected into healthy animals, caused the heart to beat more quickly and the respiration to become accelerated.² It is hardly necessary for us to consider the mechanical causes of dyspnœa, such as lessening of the calibre

¹ Marckwald : *Zeitsch. f. Biologie*, Bd. XXVI., N.F. VIII., p. 285. Marckwald, using Kronecker's method of rendering various parts of the brain inactive by plugging the arteries supplying them with paraffin injections, came to the following conclusions:—Neither the cerebrum nor the anterior part of the middle brain has a constant influence over respiration. On the other hand, if the two posterior corpora quadrigemina are paralysed, respiration changes at once, and does not regain its normal character; the long, deep, and quite regular respiratory convulsions occur, which gradually become shorter, and their depth, remaining the same, becomes regular and rhythmic. This alteration of the irregular convulsions is due to the sensory nucleus of the trigeminus. So long as this is active, the respiratory convulsions remain regular; so soon as it is paralysed, they become irregular, their stimuli proceed from the quadrigemina and from the sensory nucleus of the trigeminus which regulates the breathing after the vagi are paralysed. The posterior corpora quadrigemina have a natural tonic action, and can replace that of the vagi when these nerves are cut, while the vagi, on the other hand, can replace the removal of the corpora quadrigemina. After the removal of the posterior corpora quadrigemina the trigeminus nucleus adopts a tonic action.

² Mosso : *Die Ermüdung*, Leipzig, 1892, p. 119.

of the larynx by diphtheritic membrane, or diminution of lung space by hydrothorax, pneumothorax, empyema, pneumonia, bronchitis, or phthisis, as the diminution in the breathing space produces its effects in such an obvious manner. The pathology of spasmodic asthma is, however, by no means settled. It is quite certain that in an attack expiration is prolonged, a condition which would appear to indicate a diminution in the elasticity and contractile power of the lungs. This is also the case in breathlessness after exertion, as I have myself noticed. In asthma this condition may be due, in part at least, to a nervous affection of the contractile tissue in the lungs themselves, because that they are actively contractile was shown by Traube,¹ Ludwig and E. E. Müller,² and confirmed by Fano and Fasola.³ But it seems not improbable that another factor may be present, namely, distension of the pulmonary capillaries. So far as I know, the effect of distension of the pulmonary capillaries in dilating the air vesicles was first noticed by Hales,⁴ and was insisted on by Dr. Hensley in his Goulstonian Lectures delivered before the Royal College of Physicians in 1872. He pointed out that the network of capillaries, surrounding each pulmonary alveolus, tends, when distended with blood, to pull the walls of the alveolus apart, and thus to increase the size of the lung. The truth of Dr. Hensley's statement was experimentally proved by a German observer who imitated the conditions in the alveoli of the lung by a network of elastic tubes arranged round a bladder. The importance of this condition of lung stiffness as a cause of dyspnoea has been greatly insisted upon by Prof. von Basch,⁵ but why the blood should distend the pulmonary capillaries to such an extent, as to make them more or less rigid instead of flowing easily and quietly through them, is a question which is not easy to answer.

In a paper, read before the British Association in Bradford, September 1873,⁶ I mentioned that muscarine appeared to have the power of contracting the pulmonary capillaries and

¹ Traube: Quoted by Ludwig.

² Ludwig's *Arbeiten* for 1869, p. 38.

³ Fano and Fasola: *Archivio per le scienze mediche*, Vol. XVII., pp. 453 and 454

⁴ Hales: *Statistical Essays*, London, 1733, Vol. II., p. 77.

⁵ Von Basch: *Allgemeine Physiologie u. Pathologie d. Kreislaufs*, p. 74.

⁶ Brunton: *Reports of the British Association for 1873*.

preventing the passage of blood through them, so that, under its influence, the lungs became blanched and the right side of the heart distended.



Fig. 3. *Diagram of the normal pulmonary circulation.*

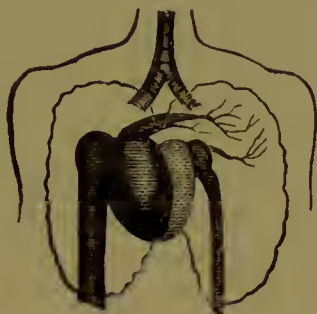


Fig 4. *Diagram of the pulmonary circulation in poisoning by muscarine.*

This effect could be immediately removed by atropine, which caused the lungs to become rosy and the right side of the heart to return to its normal condition. I attributed this to the effect of the contracting action of muscarine upon the pulmonary capillaries, an action which was antagonised by the atropine. This conclusion has been controverted by others, who have regarded this result as due to the effect of muscarine and atropine upon the heart rather than upon the pulmonary capillaries. I am still, however, inclined to believe that my opinion is correct, and that, in my experiments with muscarine, one simply had an exaggerated example of what occurs constantly during dyspnœa, where an action similar to that of muscarine is produced by the waste products contained in the venous blood. It is certain that muscarine produces great dyspnœa,¹ and that this dyspnœa can be immediately and certainly removed by atropine.² The cause of this action Schmiedeberg and Koppe were unable to explain.³ Whether the explanation I have given is correct or not, it is clear that the condition of the right heart is the same in poisoning by muscarine and in great dyspnœa.

The passage of blood through the capillaries of the lung is evidently one in which the conditions differ considerably from other parts of the body, for in experiments on artificial

¹ Schmiedeberg and Koppe: *Das Muscarin*, Leipzig, Vogel, 1869, p. 22.

² Schmiedeberg and Koppe, *Op. cit.*, p. 55.

³ *Op. cit.*, p. 55.

circulation through excised organs, such as the muscle or liver, one finds that venous blood passes rapidly through the vessels, whereas arterial blood passes slowly. As Ludwig once said to me, the organs seem to be able to regulate the supply of oxygenated blood to the tissues of which they are composed, so as not to allow combustion to go on too rapidly. But in the lungs we have completely venous blood, at the beginning of the capillary circulation, nearest to the right ventricle, and at the other end, near the left auricle, we have arterial blood. This introduces a factor of complexity which we have not in any other organ, and, whatever the cause may be, I think we may say positively that, in dyspnœa, there is a tendency to diminished pulmonary circulation, and to distension of the right side of the heart. When dyspnœa is excessive, the patient is unable to lie down, and we get the condition known as orthopnœa. Some years ago I wrote a paper in which I considered that this position was adopted by patients because in it the contraction of the diaphragm only moved the abdominal contents outwards, whereas in the recumbent position it moved them upwards.¹ I think

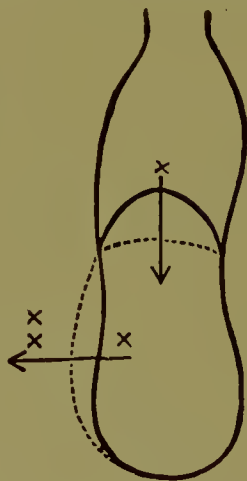


Fig. 5. *Diagram of thorax and abdomen in the upright position. The arrows show the direction in which the diaphragm and abdominal viscera move.*

that there is a certain amount of truth in this explanation, but I am convinced that it is not the whole truth, for, in going through a hospital ward, one may see patients with pneumonia,

¹ *Lancet*, July 2, 1892.

bronchitis, or phthisis, all lying down with their heads low, but those who are propped up in bed are almost always suffering from cardiac disease, and even where the primary illness is bronchitis, it is secondary cardiac dilatation which produces

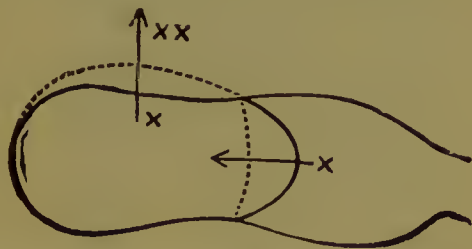


Fig. 6. *Diagram of trunk in recumbent position. The arrows show that the diaphragm moves horizontally, and lifts the viscera vertically.*

the orthopnœa. To the best of my knowledge, it was Mr. Leonard Hill, who pointed out that the relief, which the upright position afforded in such cases, was due to lessened pressure of the blood inside the right heart. One of the good old remedies, now out of fashion, for cardiac dyspnœa is blood-letting, and when I was house physician to the late Professor Hughes Bennett, I had very frequently to employ it under his direction to the great advantage of his patients. The frequency, with which he ordered it in cases of distended right heart, was all the more striking because he prided himself upon having been the man who had abolished its use in pneumonia. In many cases under his care small bleedings of 10 or 12 ozs. greatly relieved the dyspnœa in congestion of the right side of the heart, whether it was due to mitral regurgitation, or mitral obstruction, or was consequent upon chronic bronchitis. The three diseases, which I have mentioned, are the most common causes of right-sided congestion and dyspnœa. I have seen high tension in the arterial system classified in a book as a cause of cardiac dyspnœa, but I think it is incorrectly so classified, for high tension in the arteries cannot affect the lungs, so long as the aortic and mitral valves are sound. It is evident that, during the diastole, the high arterial tension can have no effect whatever upon the heart, because the column of blood in the aorta and arterial system generally is shut off completely from the heart by the closed aortic valves (Fig. 7). During the systole, when the aortic valves are open, the high tension of the blood in

the arteries presses also upon the interior of the left ventricle, but so long as the mitral valves are competent, the left auricle and lungs remain unaffected (Fig. 8). Even when the aortic

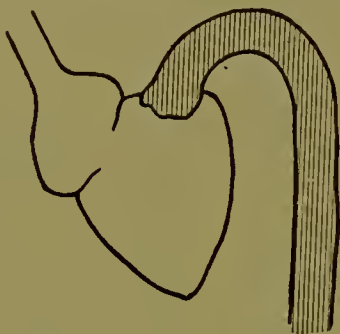


Fig. 7. *Diagram of healthy heart in diastole. The aorta is full of blood under pressure, as indicated by the shading, but the ventricle and auricle are protected from pressure by the sigmoid valves.*

valves become incompetent, the lungs remain very slightly affected, if they are affected at all, and in many cases of well-marked aortic regurgitation we find no shortness of breath, and still less do we find any dyspnoea (Fig. 8). The patients may, indeed, be absolutely unconscious that there is anything

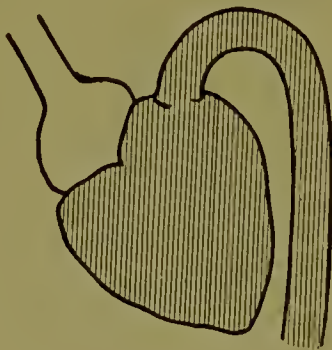


Fig. 8. *Diagram of a heart with incompetent aortic valves. The ventricle, as well as the aorta, is under pressure during diastole, but the auricle is protected by the auriculo-ventricular valves.*

the matter with them. It is when the mitral valves become affected, either by vegetations or puckering, which render them incapable of shutting completely, or in consequence of the auriculo-ventricular orifice becoming so dilated that the valves, however healthy they may be, are unable to close it, that the blood is thrown back upon the pulmonary veins at each contraction of the left (Fig. 9) ventricle, and its onward flow is thus

hindered, and dyspnœa occurs. Still worse is it when the mitral orifice is narrowed, because then, in place of the obstruc-



Fig. 9. *Diagram of a heart with incompetent aortic and mitral valves. The auricle and veins, as well as the ventricle and aorta, are under pressure constantly.*

tion being intermittent, it becomes constant, and the vessels, which will stand an intermittent strain, yield to one which is continuous.

In the early experiments made by my dear old master, Professor Ludwig, on artificial circulation through isolated parts of the body, he used blood under constant pressure.¹ He found, however, that the pressure, which had at first been sufficient to send the blood readily through the vessels of an excised organ, became insufficient to do so as the experiments went on, and it became necessary to raise it constantly. At the same time the tissues continued to undergo œdematous infiltration, and with some organs, such as the kidney, it was impossible to maintain functional activity. After a while, however, he discovered that if the pressure was rhythmically increased and lessened by raising and lowering the vessel containing the blood, at such a rate and through such a height as to imitate the natural rise and fall in the arterial pressure, which the beats of the heart caused, circulation went on for a long time without any increase in pressure being requisite, and without the appearance of œdema.²

The different effects on the respiration of distension of the right and left ventricles are well shown by some experiments of Kauders, who found, under von Basch's direction, that, when an indiarubber bag was introduced into the *left* ventricle and

¹ Ludwig's *Arbeiten* for 1868, p. 14.

² He did this about 1870, when I was working in his laboratory.

blown up so as to prevent the blood from issuing from the lung, the blood pressure in the aorta sank, the thorax became distended and moved violently, the diaphragm sank and also moved violently, so that the whole thorax was *dilated*, and respiratory movements increased. This was not due to the vagi, because the same occurrence took place when they were divided. When the *right* ventricle was obstructed in the same manner, and blood was thus prevented from entering the lungs, exactly the opposite effect on the respiration took place, the blood pressure sank, the thorax fell in, and the diaphragm rose up, so that the size of the thorax became much *diminished*. Excursions of the diaphragm, however, remained well marked.

In cases of aortic regurgitation, where, as I have already said, one frequently finds no dyspnœa, the left auricle probably plays a great part in protecting the pulmonary veins from reflex pressure. But the ventricles and auricles are not the only structures connected with the heart which have the power of rhythmical contraction. Some years ago, Sir Joseph Fayrer and I noticed that both the pulmonary veins and the vena cava possess the power of rhythmical contraction, and may continue to pulsate even when both auricles and ventricles are standing quite still.¹ This fact was known to Haller,² Senac,³ and Johannes Müller,⁴ and its importance in preventing backward flow into the veins recognised, but it had been forgotten, and was not to be found in recent books on Physiology until it was rediscovered by Fayrer and myself.

The great object we have in view in attempting to lessen cardiac dyspnœa is to facilitate the pulmonary circulation, and we will now discuss the various means at our disposal for this purpose.

The first and most efficient of all is absolute rest.

Even in healthy persons the movements of the muscles tend to raise arterial tension and to accelerate the breathing, and, if they be carried to any great extent, will cause dyspnœa. The amount of movement necessary to produce these effects

¹ Brunton and Fayrer: *Proc. Roy. Soc.*, 1874, Vol. XXII., p. 125, and *Proc. Roy. Soc.*, 1876, No. 172, p. .

² Haller: *Elementa Physiologia*, 1757, Tom. I., pp. 399 and 410, and *Mémoires sur la Nature sensible et irritable des parties du corps animal*, 1756, Tom. IV., p. 4.

³ Senac: *Traité de la structure du Cœur*, Paris, 1783, Tom. II., p. 37.

⁴ Müller's *Physiology*, translated by Baley, 2nd ed., Vol. I., p. 182.

becomes less and less as the heart fails, and at last a very slight movement indeed is sufficient to give the patient respiratory distress, as much as what would occur in healthy persons after very violent exertion. It is sometimes very difficult to make patients understand what one means by absolute rest, and unless one is careful to explain that you mean them to stay in bed, or in a chair, without moving from it *for any purpose whatever*, they will as likely as not get out of bed, and walk about the room, or even from one room to another. The rule that I lay down to my patients is: "Do absolutely nothing that any one else can do for you." If he wishes to be raised in bed, or to have the clothes adjusted, he must ask the nurse to do it for him, and more especially is it necessary to insist that the patient shall not get up to pass water or empty the bowels, but should use a urinal or bed-pan. Some patients are, however, unable to use a bed-pan, and insist upon getting up to relieve the bowels. In such cases I recommend that a stand should be made for the commode that should bring it to such a height that the patient can be moved on to it and back from it without the level of the pelvis being altered. The nature of the bed is of considerable importance, because a feather bed does not give sufficient support, and the patient can be moved much more easily on a hair mattress. In cases where the patient is absolutely unable to remain in bed, and must sit up, the same conditions must be carried out as far as possible, namely, that he must not take a single beat out of his heart that can possibly be avoided.

The next agency which is useful is massage. Ordinarily the heart has to keep up the circulation, that is, to drive the blood round through the arteries to the extremities and back again through the veins, but it is obvious that, if the masseur's hand can return the blood from the extremities through the veins, it will act the part of an assistant to the heart, and take a certain amount of work away from it. When actual dyspnœa is present, movements, however slight, are to be avoided, but when the heart is beginning to recover, and the patient is able for it, graduated movements and the Nauheim baths are most useful in aiding recovery.

Amongst drugs, the most important in its action upon the heart is digitalis, and perhaps there is no better way of giving it

than the old-fashioned pill, which contains 1 gr. digitalis, 1 gr. squill, and 1 gr. blue pill. This is the most common formula, but occasionally an additional grain of blue pill is added to it, and sometimes, as in St. Bartholomew's Hospital, some extract of hyoscyamus, where we use 2 grs. in each pill. Why the blue pill should help the action of the digitalis one cannot tell, but I think there can be no doubt whatever that it does so. Notwithstanding all the work that has been done upon the chemistry and pharmacology of digitalis, our knowledge of it is still imperfect, and I can only suspect that the different methods, in which it is used in Edinburgh and London, depend upon a different composition of the plants grown in Scotland and England. In London I believe the preparation most frequently employed is the tincture, while in Edinburgh the infusion used almost invariably to be given. When I was house physician in Edinburgh, the infusion was almost invariably given in half-ounce doses, but when I have prescribed the infusion for patients in London I have found this dose rather large, and it seemed to me more apt to produce sickness than the Scotch preparation, so that I have more commonly given it in doses of one to two drachms rather than in doses of half an ounce. I confess that I have been more inclined to use the tincture or infusion than digitaline, but nevertheless I have found Nativell's digitaline in half milligramme to milligramme doses act well. In some patients where digitalis does not succeed, strophanthus proves efficient and *vice versâ*, but I do not think one can tell beforehand which these cases are, and it is only by trial that one can find it out. One of the most valuable remedies for strengthening the heart is strychnine. Its action appears to be that of stimulating the cardiac ganglia, and in cases, where one is doubtful about giving digitalis or strophanthus, either because of their power of slowing the heart or of unduly raising the tension, one has recourse to strychnine. At the same time it proves a most useful adjunct both to digitalis and strophanthus, and may be given either by the mouth or subcutaneously.

Where the heart is failing, digitaline and strychnine may be employed subcutaneously together, and one half, even one milligramme of the former with a twentieth or even a tenth of a grain of the latter. I remember well being asked by a

practitioner, whom I met in consultation, how often I should use it in the case of an old lady with pneumonia whose heart was failing, and I said he should give the strychnine in her case until he saw the fingers jump. He followed my advice with the result that the old lady got through. Another useful remedy in dyspnœa, both of cardiac and pulmonary origin, is oxygen. I may be wrong in doing so, but I take some credit to myself for bringing both oxygen and strychnine into general use in this country. In the *Medical Record* of 1874, p. 293, I abstracted a paper on the action of strychnine on the respiratory centres, and, in a conjoint paper with Professor Cash,¹ I showed its powerful action as a cardiac stimulant. In my lectures on therapeutics for thirty years, I have insisted upon these facts. Oxygen was largely used more than a hundred years ago. Its use had fallen into abeyance except in cases of poisoning by coal gas, and it was but little used until a joint paper by Dr. Prickett and myself on its use in pneumonia² again brought it into prominence. In year books of medicine before this, I can find very few notes about its use, but, immediately after this paper, letters regarding it were frequent, and its use became general. Citrate of caffeine in doses of 2 to 5 grs., or diuretin in doses of 2 to 10 grs., every six hours, are sometimes useful adjuncts to digitalis or strophanthus. Sometimes these drugs cause irritation of the stomach or bowels, and give rise to sickness or diarrhœa which may require their discontinuance, and, as a rule, I think they do more good, if used only for a week or even less, and an interval of some days is allowed before they are given again. One of the most important means of relieving dyspnœa is certainly, I think, free purgation, and one of the best means of securing it, is by the use of compound jalap powder in doses of 20 to 60 grs. This combined with the digitalis and blue pill, already mentioned, increases the elimination of water both by the bowel and by the kidney, and lessens the congestion of the liver, which is the natural consequence of venous stagnation. It also relieves the tendency to an œdematous condition of the lung and eases the respiration. When much exudation occurs

¹ Brunton and Cash: "On the Explanation of Stannius's Experiments and on the Action of Strychnine on the Frog's Heart," *St. Bartholomew's Hospital Reports*, Vol. XVI., p. 229.

² Brunton and Prickett: *Brit. Med. Journ.*, January 23, 1892.

either into the peritoneum, or into the pleural cavities, it must of course be relieved by tapping, and sometimes the relief of dyspnœa by tapping the legs is very great. The best way of doing this depends a great deal upon the patient. Sometimes I have used with great satisfaction Southey's or Bartel's tubes, but in other cases I have found simple puncture by needles, or small incisions with plenty of absorbent wool more satisfactory. Last but not least I wish to emphasise the use of opium. For some reason or another there is quite an unnecessary fear both of this drug and of mercury, and I have seen doctors who were afraid to give mercury or calomel in a case of heart disease because there was albumen in the urine. It was with reference to the use of opium that I tried to emphasise so much the distinction between the peripheral cause and the cerebral sensation of dyspnœa. In many cases of cardiac disease the patient is prevented from falling asleep by a sudden start or a suffocative feeling, and his condition is rendered materially worse by the exhaustion consequent upon want of sleep. In cardiac dyspnœa there is no drug which will give such immediate relief as opium, and it may be administered either by the mouth, or as a subcutaneous injection of morphine, or in a way which I have not seen described, and which I think is sometimes a very satisfactory way of giving opium, viz., by mixing from half a drachm to a drachm of the tincture, not with one or two ounces of water as is usually done, but only up to two drachms with water, and injecting this amount into the rectum with an ordinary glycerine syringe. In the awful dyspnœa of renal disease there is no drug which will give relief to the same extent as opium, and these are just the cases in which the prejudice against opium, which I have already mentioned, leads men to withhold the drug. I do not know that I should have insisted upon this point, were it not that I have seen cases in which I thought that the fear of opium had led the practitioner to withhold it, as he thought, for the patient's safety, but, as I believe it, to the patient's detriment.

One other point of great practical importance I must not omit, viz., the effect of distension of the stomach by flatulence or even by food, or distension of the peritoneal cavity by ascites. All these conditions tend to tilt the heart up and embarrass its action, to say nothing of their interference

with the expansion of the lungs (Fig. 10). Food must be chosen which will not cause flatulence, and this is one advantage, though by no means the only one, of a milk diet. Carminatives

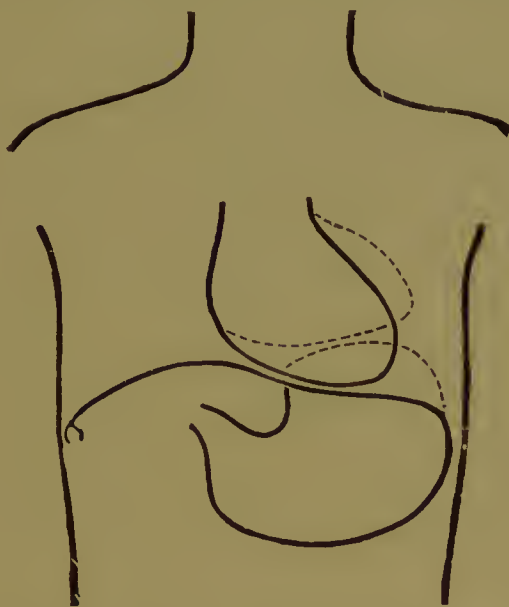


Fig. 10. *Diagram to illustrate the effect of distension of the stomach on the heart. The dark lines show the natural position of the organs. The dotted lines indicate their position when the stomach is distended.*

must be used to bring the wind away when it is present, and the relief they give is sometimes wonderful and almost instantaneous.

In order to complete this paper I ought perhaps to have discussed the action of nitrate of amyl and other vaso-dilators ; but this address is already too long, and, in conclusion, gentlemen, I have to thank you for the patience with which you have listened to it, and for the honour you have done me in asking me to give it.





